Feline Electrocution

Daniel Armstrong
Class of 2018
Clinicopathologic Conference
February 2, 2018

CPC Advisor: Dr. Marc Seitz
DVM, DABVP (Canine and Feline Practice)

INTRODUCTION

Electrocution injuries are uncommon in veterinary medicine. A database that collects patient information from 26 veterinary universities reported only 280 and 92 cases of electrical injury among dogs and cats respectively over a 35-year period.[7] Electrical injuries typically happen when young dogs or cats chew on household electrical cords. The same database showed that dogs are more commonly presented than cats and both species are typically young, with ages ranging from 2 months to 1.5 years.[9] The injuries sustained are usually from the direct effects of the electrical current and the transformation from electrical energy to heat. The severity of these injuries depend on the intensity of the current, the type of electric current (i.e. alternating versus direct), resistance to current flow through the body, and how long contact is made with the current. As expected, injury potential increases as the intensity of current increases. For example, common electrical cords found in the home are 120-volt with alternating current whereas a lightning strike can exceed 1,000,000-volts of direct current. The type of current is an important distinction because while lightening is more intense, the alternating current of electrical cords increases the risk for prolonged exposure. This is because alternating current tends to cause tetanic muscle contractions which results in the animal being unable to release the cord, resulting in longer contact with the current.[12] Direct current sources such as lightening typically does not cause muscular tetany. Resistance to current flow is also an important component of electrical injury because with more resistance comes less severe effects. Resistance typically comes from physical characteristics of the animal, with thick or dry skin providing the most resistance and moist oral mucous membranes providing little to no resistance. Less resistance allows more current to pass into the tissues, which increases the potential for widespread injury.[7]

PATHOPHYSIOLOGY

Electrical injury causes damage through three main processes: direct effects of the electrical current, thermal injury (the conversion of the current to heat in the body), and electroporation (momentary holes in cellular membranes). Mucous membranes and nerves provide the least amount of resistance compared to dry skin,[6] which is why the electrical current can readily flow through the mouth and affect electrophysiologic activity in the body. This causes a disruption in any normal neuromuscular function resulting in muscle spasms, cardiac arrhythmias, and even respiratory arrest.[7]

Thermal injuries are commonly seen with electric cord accidents and can vary in severity based on the amount of current and the length of contact time with the cord. The conversion of electrical energy to thermal energy can result in massive internal and external burns due to the superheating of intercellular and extracellular fluids, with the end result being tissue ischemia and eventual cell death.[7] Although bone has a very high resistance (as opposed to mucous membranes and nerves as mentioned above), it tends to generate a significant amount of heat which in turn causes damage to surrounding tissue.[6]

A final process that causes tissue damage and death is electroporation. Electroporation is the creation of momentary pores in cell membranes caused by the electrical current. The current is not enough to cause thermal burns to the specific area as described in the above paragraph, but will allow osmotically active proteins and other macromolecules to cross the cell membrane. Electroporation causes cell death through osmotic injury since water rapidly follows the proteins and macromolecules that enter the cell. Recognizing the potential for direct nervous system stimulation and electroporation with electrical injury may explain the association between neurogenic pulmonary edema and this form of trauma.

Neurogenic pulmonary edema is a form of non-cardiogenic pulmonary edema that is seen following an insult to the central nervous system including head trauma, severe seizures, and electrocution. Often, the onset of neurogenic edema is not clinically immediate, occurring anywhere from 1 – 36 hours after injury.[9] The actual mechanism of neurogenic pulmonary edema is not completely understood but there are two main theories concerning how it may occur. Both theories demonstrate how edema formation can occur from the direct effects of the electrical current, electroporation, or a combination of the two.

One theory hypothesizes that after an injury to the central nervous system (as is the case with electrocution, severe seizures, or head trauma), there is a large sympathetic response and catecholamine release causing severe peripheral vasoconstriction and systemic hypertension. The vasoconstriction causes an increase in afterload on the heart, reducing cardiac output, and in turn causing an increase in pulmonary venous pressure. Following Starling's Forces, this increased venous hydrostatic pressure moves fluid from the pulmonary capillaries into the lung interstitium. Hydrostatic pressure in the pulmonary vasculature is further increased by the effect of vasoconstriction on venous return. Often this fluid is a transudate with low protein content; however, a modified transudate or exudate are possible. For example, if the peripheral vasoconstriction is severe enough, endothelial pores can stretch causing injury to the endothelial cell lining, resulting in increased amounts of protein and red blood cells that will flow into the interstitial space.[12]

Another theory proposes that the mechanism of neurogenic pulmonary edema may not be related to systemic vascular pressure. Neurogenic pulmonary edema can also occur when endothelial pores increase in size and number secondary to stimulation from sympathetic nerves. These changes result in greater vascular permeability, allowing fluid, protein, and red blood cells

to travel into the interstitium.[3] This theory aligns with the concept of electroporation that occurs with electrical injury.

The actual interstitium itself may be important to the formation of pulmonary edema as well. In states of health, the interstitium helps oppose worsening transcellular fluid flux by increasing interstitial hydrostatic pressure. During the initial insult to the lungs, inflammation causes a local release of integrin, growth factors, and cytokines which break down the connective tissue of the interstitium, lowering hydrostatic pressure and increasing the ability to absorb the fluid. The net result is more fluid-flow out of the pulmonary capillaries, favoring pulmonary edema.[3] This may be a mechanism that perpetuates neurogenic pulmonary edema.

DIAGNOSTICS AND CLINICAL FINDINGS

When presented with a patient suffering from electrical injury, it is important to efficiently evaluate the patient's respiratory, cardiovascular, and nervous systems to determine if initial stabilization is necessary. This can be done with an initial triage examination. The clinician should start by noting any tachypnea, dyspnea, cyanosis, coughing, or apnea,[7] and auscultate the lungs for crackles or wheezes.[10] A cursory oral exam is performed to evaluate the airway, mucous membrane color, and capillary refill time. The presence or absence of stridor or nasopharyngeal edema should be noted. Other perfusion parameters such as heart rate, pulse quality, mentation, and temperature should be assessed. If available, pulse oximetry, blood pressure, and an electrocardiogram should be used to assess oxygenation, blood pressure, and the cardiac rhythm, respectively.[7] If upper airway obstruction or non-cardiogenic pulmonary edema are present, hypoxemia can occur. The blood pressure can be hyper- or hypotensive depending on the patient. Finally, patients may present in cardiac arrest (i.e. asystole), but will more commonly have ventricular arrhythmias noted on electrocardiogram.[7]

Once the patient is stable, a thorough physical exam is very important to assess the injuries incurred from the electrical shock. Depending on the extent of the electrocution, common findings may include singed hair or whiskers,[9] burns on the oral commissure, tongue, or palate,[7] or even dental fractures and oronasal fistulas.[10]

A focused assessment with sonography for trauma, or FAST, also aids with the initial triage assessment. Abdominal FAST scan may be unremarkable or may yield gas dilated-small intestine and decreased peristalsis secondary to ileus.[7] If non-cardiogenic pulmonary edema is present and extends to the periphery of the lung lobes, focused lung ultrasound may reveal B-lines (i.e. “lung rockets) throughout the lung fields. B-lines are vertical lines seen in the lungs that (1) extend from the plura-lung interface to the far-field, across the entire length of the screen, (2) move with the lungs as they slide across the plural surface, and (3) wash out any normally visible A-lines. These lines are actually artifacts caused by a difference in impedance between two objects, in this case air and fluid. The ultrasound beam gets trapped between these two mediums and resonates, giving the appearance of a never-ending echo. This artifact is seen with any alveolar-interstitial effusion, indicating fluid in the interstitial space and/or alveoli.[2]

Thoracic radiographs are important for diagnosing neurogenic pulmonary edema. The typical lung pattern will be alveolar to unstructured interstitial in the caudodorsal lung fields. This is in contrast to cardiogenic pulmonary edema which will be an alveolar to interstitial pattern which may start at the perihilar region and expand to be diffuse throughout the lungs.[1]

Other diagnostic procedures that are useful to help guide treatment include a complete blood count, chemistry profile, and urinalysis. Results may suggest necrosis and cell damage including hyperkalemia, myoglobinemia, myoglobinuria, hemoglobinemia, hemoglobinuria, increased lactate, and hyperglycemia.[9,7] These can occur from either muscle injury/necrosis,

ischemia, or the stress from the event. If a necropsy is performed, findings consistent with pulmonary edema (i.e. pink frothy fluid in the airways, fluid filled and congested lungs) may be seen along with sub-endocardial and sub-pericardial petechial, and gray/tan oral lesions.[9]

TREATMENT AND MANAGEMENT

Treating patients exposed to electrical injury starts with safety measures at the scene of the injury. First, it is important to turn off the source of electricity at the scene to reduce exposure to those trying to help. Once removed from the electrical source, immediate emergency evaluation (as previously described) and treatment should begin, even if the patient appears stable at presentation.[7]

Treatment for electrical injury is symptomatic as each injury is unique. These patients will often present in respiratory distress so some form of supplemental oxygen delivery is vital. This may be due to a relative obstruction from oropharyngeal edema or from a decrease of oxygen exchange in the lungs from neurogenic edema. The least stressful means of oxygen delivery should be used, often, flow-by oxygen, but in severely affected patients intubation and positive pressure ventilation may be warranted.[9]

These animals are also commonly in shock due to the relative hypovolemia so fluid boluses are usually needed.[10] It is important to note that judicious use of fluid therapy is important as aggressive fluid therapy may worsen pulmonary edema. Careful attention to the patient's respiratory quality and rate is crucial since there is no way to clinically determine the vascular integrity in the lungs. It may be useful to use a low volume fluid such as hypertonic saline or colloids, but these fluids may also worsen pulmonary edema if the vascular integrity is compromised.[7]

The severity and location of burns will affect treatment and prognosis. If possible, wounds should regularly be debrided of necrotic tissue and sucralfate can be used as an oral protectant while wounds heal.[11] The animal may not want to eat due to the oral wounds so soft foods or even a feeding tube may be needed.[9] Pain management is also important due to the burns and full body muscular contractions. An opioid should initially be used, with non-steroidal anti-inflammatory drugs only used later if the patient's condition and blood work values allow.[7]

Treatment for neurogenic pulmonary edema primarily involves supportive therapy in the form of supplemental oxygen and time. Supplemental oxygen can be provided in the form of a hood or oxygen cage. The use of a diuretic with non-cardiogenic pulmonary edema is controversial due to the pathophysiology of edema formation and inconsistent results. Diuretics usually help alleviate pulmonary edema when increased hydrostatic pressure is the sole cause, not increased vascular pulmonary permeability. Furosemide may be used cautiously as a single bolus of 0.5 – 2.0 mg/kg.[7,8,9,10,12] If a therapeutic effect is seen, diuretic therapy may be continued, but caution is advised as aggressive diuretic therapy may also worsen any preexisting hypovolemia due to excessive diuresis. Depending on the patient's oxygenation status and severity of respiratory distress, it may be necessary for some patients to remain in an oxygen cage or on a ventilator as the neurogenic pulmonary edema resolves.[7] Often if there is no underlying cardiac complications, neurogenic pulmonary edema will begin to resolve radiographically and clinically 12-24 hours after it began.[7]

Prognosis for electrocution injury is dependent on response to treatment and severity of injury, but is often fair to good. Typically, the limiting factor to recovery is the respiratory effects,[7] but severity and location of burns, as well as owner finances, may also play a roll.

CONCLUSION

In conclusion, electrical cord injury is uncommonly seen in practice but knowing the proper diagnostic modalities is vital to forming an effective treatment plan and determining prognosis. Respiratory distress is commonly seen and requires supplemental oxygen. Neurogenic pulmonary edema can be determined from the alveolar/interstitial pattern seen caudodorsally on radiographs. Treatment is often symptomatic including continued supplemental oxygen administration, an initial dose of furosemide, judicious fluid boluses, and proper wound care. Prognosis is fair to good assuming the patient's neurogenic edema resolves and wounds are appropriately managed.

REFERENCES

1. Berry CR, Grahm JP, Thrall DE. Interpretation Paradigms for the Small Animal Thorax. In: Thrall, D. E. *Textbook of Veterinary Diagnostic Radiology*. St. Louis, MO: Elsevier, Saunders. 2007: 462 – 485.

2. Doerschug KC, Schmidt GA. Intensive care ultrasound: III. Lung and pleural ultrasound for the intesivist. *Ann Am Thorac Soc* 2013; 10; 708-12.

3. Drobatz K. Pathogenesis of Neurogenic Pulmonary Edema. International Veterinary Emergency and Crital Care symposium. Nashville, TN. September 14, 2011.

4. Karatas MB, Onuk T, Güngör B, et al. Assessment of electrocardiographic parameters in patients with electrocution injury. *J of Electrocardiology* 2015; 48: 809-814.

5. Lopez A. Respiratory system. In: McGavin, D., & Zachary, J. F. *Pathologic Basis of Veterinary Disease* (4th ed.). St. Louis, MO: Elsevier Mosby. 2007: 463-558.

6. Lubin J. Electrical Injury. In: Cone D, Brice J, Delbridge J, et. al. *Emergency medical services: clinical practice and systems oversite*. Wiley and Sons. 2014; 243-244.

7. Mann FA. Electrical and Lightning Injuries. In: Silverstein, D. C. *Small animal critical care medicine*. St. Louis, MO: Elsevier, Saunders. 2015: 687 – 690.

8. Mann FA. Pulmonary Emergencies. In: Ettinger, S. J., Feldman, E. C., Cote, E. *Textbook of veterinary internal medicine: diseases of the dog and cat Volume 2, Pages 1083-2182*. St. Louis, MO: Elsevier. 2017; 443-436.

9. Mazzaferro, E. M. *Blackwell's five-minute veterinary consult clinical companion. Small Animal Emergency and Critical Care*. Ames, IA: Wiley-Blackwell. 2010: 211-216

10. McMichael M. *Handbook of Veterinary Emergency Protocols: Dog and Cat.* Jackson, WY: Teton NewMedia. 2008: 165-221.

11. Ofri R. Lens. In: Maggs, D J, Miller PE, & Ofri, R. *Slatter's fundamentals of veterinary ophthalmology*. St. Louis, MO: Elsevier. 2018: 258-276.

12. Saxon WD, Drobatz KJ, Macintire DK. Electrocution. In: Macintire, DK, and Drobatz, KJ. *Manual of small animal emergency and critical care medicine*. Chichester: Wiley-Blackwell. 2012: 493-495.

13. Shetty BS, Kanchan T, Acharya J, et al. Cardiac pathology in fatal electrocution. *Burns* 2014; 40: 45-46.